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Prognosis of Patients With Ventricular Tachycardia and Ventricular Fibrillation: Role of the Underlying Etiology

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The prognosis of 149 patients with ventricular tachycardia (n = 108) or ventricular fibrillation (n = 41) was analyzed to assess the importance of the underlying etiology of the arrhythmia. Seventy-three patients (Group I) had a previous myocardial infarction and documented late sustained monomorphic ventricular tachycardia. Thirty-five (Group II) also had a previous myocardial infarction but had late ventricular fibrillation. There were 41 patients (Group III) without coronary artery disease: 9 patients with right ventricular dysplasia, 26 with idiopathic sustained ventricular tachycardia and 6 with idiopathic ventricular fibrillation. The mean follow-up period for all patients was 22 to 57 months.

The total mortality rate in Group I (16%) and Group II (34%) and the arrhythmic mortality rate in Group I (5%) and Group II (11%) were significantly higher than the rates in Group III. In the latter group the total mortality rate was 4% for those with idiopathic ventricular tachycardia and 11% for those with right ventricular dysplasia, and there were no deaths due to arrhythmia ($p < 0.05$). Left ventricular ejection fraction was significantly lower and left ventricular end-diastolic pressure was significantly higher in Group I and Group II than in Group III. There were nonfatal recurrences of ventricular tachycardia in 33 to 56% of patients, and the number of these episodes did not differ significantly in those with and without coronary artery disease.

In summary, although the incidence of recurrence of nonfatal ventricular tachycardia is similar in patients with and without coronary artery disease, total and arrhythmic mortality is significantly higher in patients with coronary artery disease and is related to the degree of left ventricular dysfunction. The prognosis of patients with right ventricular dysplasia, idiopathic ventricular tachycardia or ventricular fibrillation is excellent when these patients are treated medically.

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The majority of patients with sustained ventricular tachycardia or ventricular fibrillation have underlying coronary artery disease and previous myocardial infarction (1–5). However, recurrent episodes of sustained ventricular tachycardia or ventricular fibrillation are also found in patients with structural heart disease due to causes other than coronary artery disease or even in patients without identifiable heart disease (6–12). The purpose of this study was to assess the importance of the underlying etiology of ventricular tachycardia or ventricular fibrillation in relation to mortality and the recurrence of the arrhythmia.

Methods

Study patients. One hundred forty-nine consecutive patients were studied. All had electrocardiographically (ECG) documented episodes of spontaneous recurrent sustained ventricular tachycardia (n = 108) or ventricular fibrillation (n = 41). There were 73 patients (Group I) with a previous myocardial infarction and sustained monomorphic ventricular tachycardia (duration >30 s), and 35 patients (Group II) with a previous myocardial infarction and ventricular fibrillation (Tables 1 and 2). There were 41 patients (Group III) without coronary artery disease: 9 had arrhythmogenic right ventricular dysplasia and 32 had no structural heart disease ("idiopathic" ventricular arrhythmias). Sustained monomorphic ventricular tachycardia was the arrhythmia in all patients with right ventricular dysplasia and in 26 of the patients without structural heart disease. Ventricular fibrillation occurred in the remaining six patients (Table 3).

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Table 1. Characteristics of 108 Patients With Coronary Artery Disease and Clinical Documented Ventricular Tachycardia (Group I) or Ventricular Fibrillation (Group II)

	CAD SMVT (%)	CAD VF (%)
Number of patients	73	35
Age (yr)	59 ± 7	
Male	64 (87.7)	30 (85.7)
Follow-up (mo)	27 ± 9	25 ± 8
Time of MI <2 mo	26 (36.1)	22 (62.9)
Arrhythmias		
2 to 6 mo	6 (8.3)	2 (5.7)
>6 mo	40 (55.6)	11 (34.4)
No. coronary VD		
1	20 (30.8)	3 (9.7)
2	20 (30.8)	6 (19.4)
3	25 (38.5)	22 (71.0)
Site of previous MI		
Anterior	31 (42.5)	20 (57.1)
Inferior	35 (47.9)	4 (11.4)
Both	7 (3.6)	11 (1.4)
EF (%)		
<40	45 (66.2)	27 (81.8)
>40	23 (33.8)	6 (18.2)
Mean	33 ± 9	32 ± 6
LVEDP (mm Hg)	17 ± 11	18 ± 8
NYHA class		
I	54 (74.0)	12 (34.3)
II	15 (20.5)	9 (25.7)
III	4 (5.5)	14 (40.0)
Exercise		
Duration (min)	6.7 ± 2.6	6.4 ± 2.8
Induced angina	9 (16.4)	4 (18.2)
Induced arrhythmia	7 (15.2)	1 (6.3)

CAD = coronary artery disease; EF = ejection fraction; LVEDP = left ventricular end-diastolic pressure; MI = myocardial infarction; No. coronary VD = number of major coronary arteries stenosed >70%; NYHA class = New York Heart Association functional class; SMVT = sustained monomorphic ventricular tachycardia; VF = ventricular fibrillation.

Diagnostic studies. All patients underwent a detailed history and physical examination, a 12 lead ECG, laboratory analysis and a chest X ray film. M-mode and two-dimensional echocardiography were carried out in 76 (70%) of the 108 patients with coronary artery disease and in all patients without structural heart disease.

Coronary angiography and left ventriculography were performed with the Judkins technique in all patients with coronary artery disease and in 38 (93%) of 41 patients with right ventricular dysplasia or idiopathic ventricular arrhythmias. All patients with right ventricular dysplasia had both left and right ventriculography.

Twenty-four hour long-term ECG monitoring was performed before angiography in all patients. One hundred seven patients were studied using a treadmill, according to the Bruce protocol. Exercise tests were symptom-limited.

Programmed electrical stimulation of the heart was performed in all patients at least once. After admission to our institution, electrophysiologic study was performed on 95 patients without antiarrhythmic drug treatment and on 100 patients receiving antiarrhythmic drugs. The stimulation protocol consisted of 1, 2 or 3 right apical ventricular premature beats given at twice the diastolic threshold during sinus rhythm and during pacing at the right ventricular apex at rates of 100, 120 and 140 beats/min. The end point of the stimulation protocol was the induction of the clinically documented ventricular arrhythmia (13).

Treatment. Among the 108 patients with coronary artery disease, amiodarone was the most commonly given drug (46 patients, 43%); class I antiarrhythmic drugs were given to 27 patients (25%) in the group. Patients with right ventricular dysplasia received amiodarone (1 patient), a class I drug (2 patients), sotalol (2 patients) and encainide (2 patients). Patients without structural heart disease were treated with amiodarone (12 patients) or sotalol (6 patients); and antiarrhythmic class I drugs were given less frequently (Table 4).

During the follow-up period, five patients (7%) with myocardial infarction and ventricular tachycardia underwent aortocoronary bypass grafting and four patients (6%) had aneurysmectomy with endocardial resection. Among patients with myocardial infarction and ventricular fibrillation, three (9%) had aortocoronary bypass grafting and two (6%) underwent aneurysmectomy, one having additional endocardial resection. One patient with right ventricular dysplasia underwent surgery (right ventricular disconnection) and died postoperatively from heart failure. Among patients with idiopathic ventricular tachycardia, an antitachycardia pacemaker was implanted in three patients (12%) and endocardial resection was performed in one patient (3.8%). One patient with idiopathic ventricular fibrillation and recurrent episodes of ventricular fibrillation received an automatic implantable defibrillator.

Follow-up. All patients were seen in the outpatient clinic at least once a year. The mean follow-up periods of the different groups are listed in Tables 1 and 2. All cases of death were verified and classified by interviews with relatives, family or hospital physicians and, if possible, by analyzing hospital charts. Sudden death in this patient population was defined as witnessed death occurring by ≤1 h of the onset of symptoms.

Statistical analysis. Statistical analysis was performed using the paired and unpaired Student's *t* test and the chi-square test. Survival curves were analyzed using life-table analysis. P values <0.05 were considered significant.

Results

Total mortality and incidence of sudden death. During the follow-up period of 39 months, 26 (17%) of the 149 patients died, 8 (5%) suddenly. Among the 73 patients with a previous myocardial infarction and ventricular tachycardia

Table 2. Characteristics of 20 Patients With Coronary Artery Disease Who Died Suddenly or of a Cardiac Cause Compared With 84 Survivors

	CAD SMVT					
	SD (%)	CD (%)	SV (%)	SD (%)	CD (%)	SV (%)
Number of patients	4	7	61	4	5	23
Age (yr)	57 ± 8	57 ± 7	61 ± 8	56 ± 7	58 ± 10	57 ± 9
Male	4 (100)	7 (100)	52 (81)	3 (75)	5 (100)	19 (83)
Time from MI <2 mo	3 (75)	3 (43)	21 (34)	2 (50)	3 (60)	14 (61)
Arrhythmia						
2 to 6 mo	—	1 (14)	4 (7)	—	—	2 (9)
>6 mo	1 (25)	3 (43)	36 (59)	2 (50)	2 (40)	7 (30)
No. coronary VD						
1	1 (25)	1 (14)	20 (33)	—	—	4 (17)
2	1 (25)	1 (14)	20 (33)	1 (25)	1 (20)	5 (22)
3	2 (50)	5 (71)	21 (34)	3 (75)	4 (80)	14 (61)
Site of previous MI						
Anterior	1 (25)	5 (71)	25 (41)	2 (50)	2 (40)	15 (65)
Inferior	2 (50)	2 (29)	30 (49)	1 (25)	—	3 (13)
Both	1 (25)	—	6 (10)	1 (25)	3 (60)	5 (22)
EF (%)						
<40	4 (100)	5 (71)	38 (62)	4 (100)	4 (80)	17 (74)
>40	—	2 (29)	23 (38)	—	1 (20)	6 (26)
Mean	31 ± 7	34 ± 9	55 ± 12	32 ± 5	32 ± 8	31 ± 9
LVEDP (mm Hg)	21 ± 17	17 ± 10	17 ± 9	18 ± 4	19 ± 4	20 ± 11
NYHA class						
I	—	2 (29)	45 (74)	1 (25)	1 (20)	9 (39)
II	3 (75)	—	13 (21)	1 (25)	—	7 (30)
III	1 (25)	5 (71)	3 (5)	2 (50)	4 (80)	7 (30)
Exercise						
Duration (min)	5 ± 2	6 ± 2	7 ± 3	7 ± 2	7 ± 2	6 ± 3
Induced angina	—	—	9 (15)	—	—	4 (17)
Induced arrhythmia	2 (50)	2 (29)	3 (15)	—	—	1 (4)

CD = cardiac death; SD = sudden cardiac death; SV = survivors; other abbreviations as in Table 1.

(Group I), 12 (16%) died, 4 suddenly, 4 from recurrent myocardial infarction, 3 from congestive heart failure and 1 from suicide. Among the 35 patients with a previous myocardial infarction and ventricular fibrillation (Group II), 12 (34%) died, 4 suddenly, 3 from recurrent myocardial infarction, 3 from congestive heart failure, 1 from a stroke and 1 from suicide. Of the 41 Group III patients, no patient died suddenly and one patient died from low cardiac output after surgery. Among patients without structural heart disease, no patient died suddenly, and one died from lung cancer.

Total mortality was significantly higher in patients with coronary artery disease (24 [29%] of 108) than in those without left ventricular disease (2 [5%] of 41) ($p < 0.02$). In addition, cardiac mortality was higher in patients with coronary artery disease and ventricular fibrillation (10 [29%] of 35) than it was in patients with coronary artery disease and ventricular tachycardia (11 [15%] of 73) ($p = 0.08$) (Fig. 1). No significant differences were found between the incidence of sudden death in patients with coronary artery disease and that in patients with ventricular tachycardia (Group I) (4 [6%] of 73) or ventricular fibrillation (Group II) (4 [11%] of 35) (Fig. 2).

Previous clinical characteristics. All patients with coronary artery disease had a history of myocardial infarction and showed typical ECG signs (Q waves). Among the 73 patients with sustained monomorphic ventricular tachycardia (Group I), the prior infarction was anterior in 31 (43%), inferior in 35 (48%) and both anterior and inferior in 7 (10%); among the 35 patients with ventricular fibrillation (Group II), the prior infarction was anterior in 20 (57%), inferior in 4 (11%) and both anterior and inferior in 11 (31%) (Table 1).

The interval between myocardial infarction and ventricular tachycardia was >6 months in 40 patients (56%), whereas it was <2 months in most (63%) of the 35 patients with ventricular fibrillation.

Functional class. Of the 73 Group I patients with a previous myocardial infarction and ventricular tachycardia, 57 (74%) were classified in the New York Heart Association functional class I, 15 (21%) in class II and 4 (6%) in class III. In contrast, among the 35 patients with a prior infarction and

Table 3. Characteristics of Group III Patients With Arrhythmogenic Right Ventricular Dysplasia, Idiopathic Ventricular Tachycardia and Idiopathic Ventricular Fibrillation

	RVD (n = 9)	Idio SMVT (n = 26)	Idio VF (n = 6)
Age (yr)	31 ± 7	35 ± 15	37 ± 7
Male	8 (89%)	16 (62%)	6 (100%)
Follow-up (mo)	42 ± 12	57 ± 14	47 ± 9
EF %			
<40	—	—	—
>40	9 (100%)	26 (100%)	6 (100%)
Mean	63 ± 6	64 ± 6	64.0 ± 8
LVEDP (mm Hg)	5 ± 1	8 ± 5	7 ± 3
NYHA class I	9 (100%)	26 (100%)	6 (100%)
Exercise			
Duration (min)	10.3 ± 3.9	10.6 ± 2.9	11.8 ± 4.1
Arrhythmia	4 (57%)	5 (36%)	1 (17%)

Idio = idiopathic, RVD = right ventricular dysplasia; other abbreviations as in Table 1.

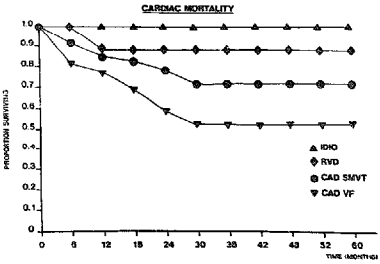

Figure 1. Cardiac mortality in relation to the underlying heart disease. CAD = coronary artery disease; IDIO = idiopathic; RVD = right ventricular dysplasia; SMVT = sustained monomorphic ventricular tachycardia; VF = ventricular fibrillation.

ventricular fibrillation (Group II), 12 (34%) were classified in functional class I, 9 (26%) in class II and 14 (40%) in class III.

Most of the patients who died were in functional class II or III (Group I: 6 of 11 patients, 55%; Group II patients: 7 of 9 patients, 78%) (p = ns). All patients with no coronary artery disease (Group III) had functional class I heart failure (Table 3).

Left ventricular function and coronary angiography. Significant differences were found between degree of left ventricular dysfunction in patients with and without coronary artery disease (Tables 1 to 3). Left ventricular ejection fraction was significantly higher and left ventricular end-diastolic pressure was significantly lower in patients without coronary artery disease (Group III) ($p < 0.05$). Ejection fraction and left ventricular end-diastolic pressure did not differ significantly in patients with myocardial infarction who died suddenly and patients who died from a cardiac cause or in patients who died suddenly and survivors (Table 2). However, there was a trend toward a lower ejection fraction and a higher left ventricular end-diastolic pressure in patients who died suddenly (mean ejection fraction 35 ± 7%, mean end-diastolic pressure 21 ± 17 mm Hg) compared with values in survivors (mean ejection fraction 35 ± 12%, mean end-diastolic pressure 17 ± 9 mm Hg). Three vessel coronary artery disease was significantly ($p < 0.01$) more prevalent in the group with ventricular fibrillation (22 patients 71%) than in the group with ventricular tachycardia (25 patients, 39%). In addition, 3 patients (10%) with ventricular fibrillation had only one vessel disease compared with 20 patients (31%) with ventricular tachycardia ($p < 0.05$). Patients who died had a greater number of coronary arteries involved than did survivors (Table 2).

Long-term electrocardiographic monitoring. There were no significant differences between the incidence of isolated premature ventricular beats (Lown class 1 to 3) or "complex" ventricular arrhythmias (Lown class 4 and 5) in

Table 4. Antiarrhythmic Drug Treatment in Patients With Coronary and Noncoronary Artery Disease

	CAD SMVT (%)	CAD VF (%)	RVD (%)	Idio SMVT (%)	Idio VF (%)
Number of patients	73	35	9	26	6
Amiodarone	28 (38)	18 (51)	1 (11)	9 (35)	3 (50)
Propafenone	14 (19)	2 (6)	—	4 (15)	—
Flecainide	8 (11)	2 (6)	—	2 (8)	—
Bepridil	5 (7)	1 (3)	2 (22)	1 (4)	—
Mexiletine	1 (1)	1 (3)	—	1 (4)	—
Disopyramide	—	—	—	2 (8)	—
D-sotalol	5 (7)	1 (3)	2 (22)	6 (23)	—
Procainamide	6 (8)	5 (14)	—	—	—
Quinidine	—	1 (3)	—	1 (4)	1 (17)
Other	4 (6)	2 (6)	2 (22)	—	2 (33)

Abbreviations as in Tables 1 and 3.

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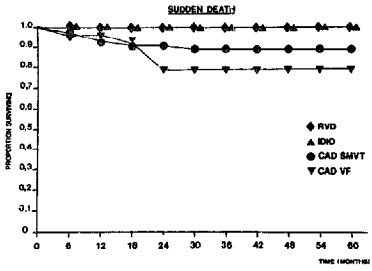

Figure 2. Incidence of sudden death in relation to the underlying heart disease. Abbreviations as in Figure 1.

patients with myocardial infarction and ventricular tachycardia or fibrillation and that in patients without coronary artery disease. However, the incidence of complex arrhythmias was >50% in all groups.

Ambulatory ECG recordings during antiarrhythmic drug treatment showed a 60% incidence rate of class 1 to 3 and a 40% incidence rate of class 4 arrhythmia in patients with myocardial infarction and ventricular tachycardia or ventricular fibrillation. The incidence rate of class 1 to 3 arrhythmia was 30% and that of class 4 arrhythmia was 60% in patients without coronary artery disease (Table 5).

Programmed stimulation in absence of antiarrhythmic drugs (Table 6). Sustained monomorphic ventricular tachycardia was induced in 35 (83%) of 42 patients with coronary artery disease and spontaneous ventricular tachycardia (Group I). In five Group I patients (12%) nonsustained ventricular tachycardia was inducible and in the remaining two patients (5%) no arrhythmias were inducible. In patients with spontaneous ventricular fibrillation (Group II), ventricular fibrillation was induced in 6 (32%) of 19 patients, and sustained monomorphic ventricular tachycardia in 10 (53%); in the remaining 3 patients (16%) no or only nonsustained ventricular tachycardia was induced.

During electrophysiologic study in Group III, sustained ventricular tachycardia was induced in all patients with right ventricular dysplasia. In contrast, among 21 patients with idiopathic ventricular tachycardia, sustained ventricular tachycardia was induced in 9 (43%) and no or nonsustained ventricular tachycardia was observed in 12 (57%). In four patients with idiopathic ventricular fibrillation programmed stimulation was performed; sustained monomorphic ventricular tachycardia was induced in two patients and ventricular fibrillation in the remaining two.

The mode of initiation of ventricular arrhythmias was not significantly different in patients with and without myocardial infarction. In the majority of patients, two ventricular extrastimuli were required to induce the arrhythmia (Table 6).

Programmed stimulation during antiarrhythmic drug treatment (Table 2). Among 50 patients with myocardial infarction and ventricular tachycardia (Group I), sustained monomorphic ventricular tachycardia was inducible in 40 (80%), ventricular fibrillation in 1 (2%) and nonsustained ventricular tachycardia in 5 (10%); no arrhythmias were induced in 4 patients (8%). Among 28 patients with myocardial infarction and initial ventricular fibrillation (Group II), ventricular fibrillation was induced in none, sustained monomorphic ventricular tachycardia in 12 (43%) and nonsustained ventricular tachycardia in 2 (7%); no arrhythmias were induced in 8 patients (29%).

Among seven patients with right ventricular dysplasia,

Table 5. Incidence of Spontaneous Ventricular Arrhythmias Detected by 24 h Long-term ECG Recordings

	CAD SMVT (%) (n = 73)	CAD VF (%) (n = 35)	RVD (%) (n = 9)	Idio SMVT (%) (n = 26)	Idio VF (%) (n = 6)
A. Without antiarrhythmic treatment					
Number of patients	47 (64)	17 (49)	6 (67)	17 (65)	2 (33)
Lown class					
0-2	14 (30)	5 (29)	—	2 (12)	1 (50)
3	3 (6)	—	—	3 (18)	—
4a	4 (9)	—	3 (50)	2 (12)	—
4b	26 (55)	12 (71)	3 (50)	10 (59)	1 (50)
B. With antiarrhythmic drug treatment					
Number of patients	50 (69)	20 (57)	3 (33)	11 (42)	7 (67)
Lown class					
0-2	25 (50)	12 (60)	—	2 (18)	2 (50)
3	5 (10)	—	1 (33)	2 (18)	—
4a	3 (17)	2 (10)	1 (33)	1 (9)	—
4b	17 (34)	6 (30)	1 (33)	6 (55)	2 (50)

Abbreviations as in Tables 1 and 3.

Table 6. Incidence of Inducibility During Programmed Stimulation Without Antiarrhythmic Drug Treatment in 149 Patients With and Without Coronary Artery Disease

	CAD SMVT (n = 73) (%)	CAD VF (n = 35) (%)	RVD (n = 9) (%)	Idio SMVT (n = 26) (%)	Idio VF (n = 6) (%)
EP study performed	42 (58)	19 (54)	9 (100)	21 (81)	4 (67)
VF	—	6 (32)	—	—	2 (50)
SMVT	35 (83)	10 (53)	9 (100)	9 (43)	2 (50)
NSVT	5 (12)	2 (11)	—	6 (29)	—
NI	2 (5)	1 (5)	—	6 (29)	—
Stimuli					
1 ES	10 (25)	6 (33)	2 (25)	5 (33)	1 (25)
2 ES	22 (55)	10 (56)	4 (50)	8 (53)	1 (25)
3 ES	8 (20)	2 (11)	2 (25)	2 (13)	2 (50)
Cycle length clinical VT (ms)	823 ± 57	—	277 ± 31	326 ± 67	—
Cycle length-induced SMVT	320 ± 96	307 ± 78	270 ± 35	326 ± 61	350 ± 14

EP = electrophysiologic study; ES = extrastimuli; NI = noninducible; NSVT = nonsustained ventricular tachycardia; other abbreviations as in Table 1.

sustained monomorphic ventricular tachycardia was induced in six patients and nonsustained ventricular tachycardia in one patient.

Among 11 patients with idiopathic ventricular tachycardia, sustained monomorphic ventricular tachycardia was still inducible in 3 patients (27%), ventricular fibrillation in 1 patient (9%) and nonsustained ventricular tachycardia in 2 patients (18%); no arrhythmias were induced in 5 patients (46%). Electrophysiologic study with the patients receiving drugs was performed on four patients with spontaneous idiopathic ventricular fibrillation: ventricular fibrillation was induced in one patient, sustained monomorphic ventricular tachycardia in one, nonsustained ventricular tachycardia in one and no arrhythmias in one.

The mode of initiation did not differ significantly in patients with and without myocardial infarction. In the majority of patients, two ventricular extrastimuli were required to induce the arrhythmia.

Ventricular tachycardia cycle length. The cycle length of the clinical documented ventricular tachycardia and the induced sustained ventricular tachycardia before or during antiarrhythmic drug treatment showed no significant differences between patients with and without coronary artery disease (Tables 6 and 7). However, both the cycle length of the clinical ventricular tachycardia and the induced ventricular tachycardia in the absence of drugs were shorter in patients with right ventricular dysplasia compared with the other patients. No significant differences were found in the cycle length of induced ventricular tachycardia in patients who were or were not receiving antiarrhythmic drug treatment.

Recurrences of ventricular tachycardia. Recurrences of arrhythmic events were fatal only in patients with coronary artery disease (Groups I and II). Four patients (6%) with spontaneous sustained monomorphic ventricular tachycardia died suddenly, as did four patients (11%) with documented spontaneous ventricular fibrillation.

The incidence of nonfatal arrhythmic events during the follow-up did not differ in patients with and without previous myocardial infarction. Nonfatal arrhythmic events occurred in 19 patients (26%) in Group I (prior infarction and ventricular tachycardia) and in 2 patients (6%) in Group II (prior

Table 7. Incidence of Inducibility During Programmed Stimulation With Antiarrhythmic Drug Treatment in 149 Patients With and Without Coronary Artery Disease

	CAD SMVT (n = 73) (%)	CAD VF (n = 35) (%)	RVD (n = 9) (%)	Idio SMVT (n = 26) (%)	Idio VF (n = 6) (%)
EP study performed	50 (69)	28 (80)	7 (78)	11 (42)	4 (67)
VF	1 (2)	—	1 (9)	1 (25)	1 (25)
SMVT	40 (80)	12 (43)	6 (86)	3 (27)	1 (25)
NSVT	5 (10)	2 (7)	1 (14)	2 (18)	1 (25)
NI	4 (8)	8 (29)	—	5 (46)	1 (25)
Stimuli					
1 ES	7 (15)	4 (29)	1 (17)	2 (33)	1 (33)
2 ES	30 (65)	8 (57)	4 (67)	3 (50)	2 (67)
3 ES	9 (20)	2 (14)	1 (17)	1 (17)	—
Cycle length (ms)	370 ± 57	320 ± 68	330 ± 59	308 ± 111	360 ± 96

Abbreviations as in Tables 1 and 6.

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infarction and ventricular fibrillation). The incidence of nonfatal arrhythmic events was higher in patients with noncoronary artery disease (Group III). Five patients (56%) with right ventricular dysplasia, 10 patients (39%) with idiopathic ventricular tachycardia and 2 patients (33%) with idiopathic ventricular fibrillation had recurrent nonfatal ventricular tachycardia.

Antiarrhythmic drug treatment and clinical outcome. Four patients in Group I (prior infarction and spontaneous sustained ventricular tachycardia) had a fatal arrhythmic event. Two of them were receiving amiodarone (200 mg/day and 400 mg/day, respectively) at the time of sudden death; the remaining two patients were treated with bepridil (400 mg/day). Whether a proarrhythmic effect was the cause of sudden death is unknown to us.

In Group II (prior infarction and initial ventricular fibrillation), four patients died suddenly. At the time of death, two patients were receiving amiodarone (400 mg/day), one patient was receiving flecainide (200 mg/day) and one was receiving procainamide (3,000 mg/day).

Nonfatal episodes of ventricular tachycardia recurred in 49 patients (29%), in the majority during treatment with a class I antiarrhythmic drug. Approximately 40% of the patients in Groups I and II with recurrent ventricular tachycardia were receiving amiodarone, compared with 20% of patients in Group III with recurrent ventricular tachycardia.

Discussion

Identification of patients at high risk for sudden death remains a difficult problem. It has been reported that the etiology of ventricular tachycardia is an important consideration in treatment and prognosis (14). Therefore, we evaluated the incidence of sudden death in relation to the underlying heart disease.

Prognosis in patients without coronary artery disease. There is general agreement that patients with sustained ventricular tachycardia unrelated to coronary artery disease have a better prognosis than do patients with ventricular tachycardia after myocardial infarction (15). In our series of 26 patients with sustained ventricular tachycardia without structural heart disease, no patient died suddenly during a mean follow-up period of 47 months. In addition, no sudden death occurred in patients with "idiopathic" ventricular fibrillation. These data are in agreement with previous studies demonstrating an excellent prognosis in such patients (16–19). However, there are some reports of sudden death in patients with idiopathic ventricular tachycardia (20–22).

Prognosis in patients with coronary artery disease. The incidence of sudden death is much higher in patients with coronary artery disease (23–25), particularly in patients having both poor left ventricular function and residual ischemia (26,27). The prognosis of patients with coronary artery disease is clearly influenced by the presence of ventricular arrhythmias (28). Risk stratification in these groups of patients is essential for treatment and prognosis. In this study, the total mortality rate in patients with coronary artery disease and spontaneous ventricular tachycardia or ventricular fibrillation was 16.4% and 34.3%, respectively. These data are in agreement with those of other investigators (29–31).

Role of left ventricular dysfunction. Total mortality and incidence of sudden death were clearly related to the degree of left ventricular dysfunction. Left ventricular damage was more extensive in patients who died during the follow-up period than in survivors. The number of involved coronary arteries, left ventricular ejection fraction and clinical data (exercise test, functional class) differed markedly in patients with and without coronary artery disease. In addition, both clinical and angiographic and hemodynamic findings were significantly different in patients with and those without coronary artery disease.

Role of ambulatory ECG monitoring and programmed stimulation. Therefore, to evaluate the risk of sudden death, clinical information about the underlying heart disease, noninvasive or invasive studies, or both, are required. Long-term ambulatory ECG monitoring is a widely accepted method, in the diagnostic evaluation of ventricular arrhythmias, to determine the frequency and severity of ventricular ectopic rhythm (32). In our study it was not possible to stratify the risk of a patient to develop malignant arrhythmias using these criteria alone. Classification of a patient at high risk for sudden death was not possible on the basis of programmed stimulation alone, a finding in agreement with other reports (4,33–40).

Role of antiarrhythmic drug therapy. In patients with and without coronary artery disease, the incidence of nonfatal arrhythmic events during antiarrhythmic drug treatment did not differ significantly and did not allow for a risk stratification. Although antiarrhythmic drug treatment frequently could not prevent recurrent ventricular tachycardia, a recurrent arrhythmia was rarely associated with sudden death in all groups.

Limitations. Our patients with idiopathic ventricular tachycardia did not undergo endomyocardial biopsy; therefore, myocarditis, as reported by Vignola et al. (41), was not completely excluded. We had no clinical reason to suspect myocarditis in any patient classified as having "idiopathic" ventricular tachycardia. As reported by Vignola et al. (42), however, clinically silent lymphocytic myocarditis could have been responsible for ventricular tachycardia in some of our patients. Also, data from programmed stimulation and long-term ECG monitoring were not available for all patients. Conclusions in terms of predicting recurrences of tachycardias apply only to patients for whom data were available.

Clinical implications. The risk of sudden death in patients with sustained monomorphic ventricular tachycardia or ventricular fibrillation is clearly related to the underlying heart disease, particularly to the degree of left ventricular dysfunction. Patients without structural heart disease or those with right ventricular dysplasia have an excellent prognosis when treated medically, despite recurrences of ventricular tachycardia. Prognosis, however, may differ in patients whose ventricular tachycardia is associated with types of heart disease, cardiomyopathy for example, other than those included in this study.

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